gradually decreased over a period of approximately two weeks towards an OR of one (2).

The possible contribution of airway infections, which occur more frequently in cold winter months than in summer, have not been listed by Spencer et al. (1) among the possible explanations for the higher incidence of AMIs in winter time. Evidence from smaller hospital-based studies has already previously indicated that an association between acute chest infections and AMI might exist (3–5). The pathophysiologic mechanism of such an association is speculative and could involve changes in circulating clotting factors, an increased risk for acute rupture of arteriosclerotic plaque during a chest infection, or a variety of other mechanisms. The role of acute and chronic infections in the etiology of coronary heart disease and AMI needs to be better understood; it might be one of the keys towards an explanation why more AMIs occur in winter than in summer.

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Anticoagulation in Dilated Cardiomyopathy

We read with interest the recent review by Koniaris et al. (1) concerning anticoagulation in dilated cardiomyopathy. Evidence from published studies does not convincingly demonstrate the benefits of anticoagulation in patients with dilated cardiomyopathy. As described by Koniaris et al., (1) a prospective, randomized clinical trial of long-term anticoagulation in patients with dilated cardiomyopathy is feasible. However, studies employing hemostatic markers indicating a biochemical imbalance between procoagulant and anticoagulant mechanisms in the blood may be useful to evaluate the appearance of thrombotic phenomena in these patients (2). By measuring plasma levels of hemostatic markers, we previously found that, in patients with dilated cardiomyopathy, plasma levels of fibrinopeptide A and thrombin-antithrombin III complex, markers of coagulation activation and thrombin generation, were significantly higher than those in normal subjects (3). Their levels showed a positive correlation with left ventricular end-diastolic volume and a negative correlation with fractional shortening of the left ventricle. Although Koniaris et al. (1) suggested that aspirin monotherapy may be beneficial for risk reduction of thromboembolism in patients with dilated cardiomyopathy, plasma levels of platelet factor-4 and beta-thromboglobulin, markers of platelet activation, were not elevated in these patients compared with normal subjects. In addition, we previously observed that aspirin monotherapy suppresses platelet function, but does not affect coagulation activity (4). These findings support the premise that anticoagulant therapy, rather than antiplatelet therapy, is more effective for the prevention of systemic embolism in patients with dilated cardiomyopathy, particularly in those with severe left ventricular enlargement and dysfunction.

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